LETTERS TO THE EDITOR

Neonatal respiratory distress syndrome

EDITOR,—The report of the working group on the management of neonatal respiratory distress syndrome states that 'normal' limits for blood gas variables cannot be stated, and appropriate levels of arterial oxygen saturation (Sao_2) have not yet been agreed.[1] As a result they recommend that further research includes determining safe levels of Sao_2 in infants with respiratory distress syndrome and chronic lung disease. Despite this area of uncertainty, they state that 'the recommended range for Pao_2 [arterial oxygen tension] is 6–10 kPa and that acceptable levels for Sao_2 of 85–93% have been proposed'. These ranges are stated without reference to reported normal values and may in fact be detrimental to an infant.

The fifth centile for levels of Sao_2 in 'healthy' preterm infants without lung disease and either ready for discharge from the neonatal unit,[2] or in the first week of life,[3] are 95·7% and 95·5% respectively. These measurements were made with the Nellcor N100/N200 during quiet sleep and excluding the transient drops in Sao_2 after normal apnoeic pauses. By definition, hypoxaemia is below these levels and should be corrected when treating respiratory failure.

In order to avoid hyperoxaemia (for which definitions vary) in preterm infants receiving mechanical ventilation, the upper limit of Sao_2 must also be specified. Southall *et al*, using the Nellcor N100 pulse oximeter, found from 169 measurements in 81 patients, 24 occasions when Pao_2 was $\geq$100 mm Hg ($\geq$13·3 kPa): the Sao_2 was >97% on 23 occasions and 95% on one.[4] Bucher *et al*, comparing the Nellcor N100 and the Ohmeda Biox pulse oximeters, reported that the Nellcor N100 identified hyperoxaemia (Pao_2 >90 mm Hg or >12·0 kPa) with 100% sensitivity if an alarm level of 95% was chosen.[5] The Ohmeda Biox 3700 pulse oximeter, however, had a sensitivity of only 37% at this alarm level. A more recent study using the Ohmeda Biox 3700 found that the lower limb Sao_2 levels of 95–96% would result in a postductal Pao_2 of $\leq$99 mm Hg ($\leq$13·2 kPa) for 95% of the time.[6] Another study, involving 137 hyperoxaemic instances (defined as Pao_2 >80 mm Hg or 10·7 kPa) in 50 patients, found that the Nellcor N200, with the alarm limit set at 95%, identified 95% of these instances.[7] The highest Pao_2 value not identified by the pulse oximeter was 104·5 mm Hg (13·9 kPa).

Thus, when using the Nellcor pulse oximeter, hyperoxaemia may only be avoided with sufficient certainty if the upper alarm limit is kept at 95–97%. This implies that Sao_2 values above 95–97%, although obviously 'normal' for healthy preterm neonates, cannot be recommended for preterm infants receiving respiratory support. Because of this unfortunate overlap between normal Sao_2 levels, and those that may be associated with a 'dangerously' high Pao_2 we would recommend that baseline Sao_2 is kept between 94 and 96% in preterm infants receiving additional inspired oxygen and monitored using the Nellcor pulse oximeter. In addition, and as the working group stresses, the monitoring of arterial line Po_2 values will remain essential to assess the effect of respiratory support and to be certain of avoiding hyperoxaemia.

Lower levels of Sao_2 may be detrimental: firstly, preterm infants with a low baseline Sao_2 desaturate further with apnoeic pauses than those who are adequately oxygenated.[8] Secondly, hypoxaemia as a result of lung hypoxia increases both pulmonary vascular and bronchiolar smooth muscle tone.[9 10] Such changes may prolong ventilatory and oxygen dependence, increase the risk for severe hypoxaemic episodes, and result in infants being treated with diuretics and bronchodilators. Inspired oxygen is a potent pulmonary vasodilator and may also prevent bronchoconstriction.[9 10]

We agree with the recommendation in appendix A that one of the most important steps forward here would be a randomised controlled trial looking at the levels of oxygenation that should be aimed for in treating neonates with respiratory distress syndrome. Such a study should include information concerning retinopathy as well as major outcome variables, such as death, chronic lung disease, the duration of inspired oxygen, and levels of required ventilation.

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Varicella zoster virus infection in pregnancy

EDITOR,—The recent annotation describes the possible effects on the fetus from maternal varicella zoster infection, including the embryopathic effects of first trimester infection.[1] The authors quote Alkalay *et al*[2] as suggesting, in their review of all published reports of the fetal varicella syndrome (FVS), that the presence of cicatricial skin lesions corresponding to a dermatome distribution is essential. We would disagree, as one well documented case had other features of FVS but no skin lesions,[3] and we have recently seen a similar case.

A baby boy was born at 36 weeks' gestation, with a birth weight of 1400 g, well below the third centile. His mother had had chicken pox at 16 weeks. At birth, apart from being severely growth retarded, there were no abnormal external features. No anti-varicella zoster IgM was detectable in his blood, and this, together with the absence of cicatricial skin lesions or limb abnormalities, reassured us that he had probably escaped the FVS. However, further examination revealed severe chorioretinitis, and at 10 weeks, while still on the neonatal unit, he developed a typical shingles rash in the C6 dermatome distribution. There had been no postnatal contact with anyone with active chicken pox or shingles. Electron microscopy on fluid aspirated from the vesicles identified varicella zoster particles. There was no serological evidence of any other congenital infection. He has exhibited several other features well described in the FVS,[2] including severe gastro-oesophageal reflux and bulbar palsy resulting in several near fatal episodes of aspiration pneumonia, cortical atrophy, profound developmental delay, and a hoarse, weak cry,[4] but he has no skeletal or urinary tract abnormalities. Contrast radiological studies have shown almost complete absence of oesophageal and gastric peristalsis, and he is fed via a jejunostomy tube. He is unable to swallow saliva and has permanent respiratory signs and symptoms due to aspiration.

We are confident that our patient does have FVS, but were misled by the absence of skin lesions and limb abnormalities at birth. It is clear that babies may be born after maternal varicella zoster infection up to 20 weeks' gestation[2] who are severely affected by varicella embryopathy, but in whom the external appearances may be reassuringly normal. It is, therefore, very important to examine such infants closely for other features of FVS.

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